SUBJECTS WITH ESTABLISHED KNEE OSTEOARTHRITIS PRESENT ELEVATED COMPRESSIVE KNEE CONTACT FORCES BUT MINIMISE MEDIAL SHEAR FORCES DURING GAIT

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INTRODUCTION

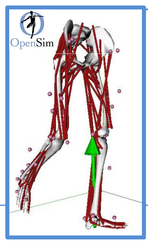
Osteoarthritis (OA) is a chronic degenerative joint disease that most frequently affects the knee. Patients complain about pain, muscle weakness, stiffness and instability. This may ultimately lead to a loss of independence, reduced quality of life and high health-related costs[1]. Aberrant knee joint loading has been identified as a potential factor affecting onset and progression of knee OA. The aim of this study is to estimate if knee joint reaction forces (KCF) during gait differ between healthy adults and subjects with increasing severity of knee OA using subject-specific musculoskeletal simulations. KCF reflect the combined effect of intersegmental and musculotendon forces. We hypothesize that subjects with increasing levels of OA involvement, the KCF are significantly altered due to the combined effect of aberrant movement dynamics, muscle coordination and joint geometry.

METHODS

Fifty-nine patients (women, mean age of 65.0) were recruited and separated into three groups:

1. **Control subjects** (n=20) – **C0** – asymptomatic, had no history of knee OA or other pathology;
2. **Early medial knee OA** (n=16) – **OA1** – based on novel classification criteria by Luyten et al.[2]: knee pain, a K&L grade 0, 1 or 2$^-$ and structural changes observed by MRI;
3. **Established medial knee OA** (n=23) – **OA2** – based on slightly adapted ACR classification criteria[3]: knee pain, age above 50, stiffness <30 min and crepitus, presence of minimum grade 2$^+$ on K&L scale.

▪ Active 3D motion analysis system (Krypton, Metris);
▪ Force plate (Bertec Corporation),



3D model
▪ 24 DoF;
▪ 2 DoF Tibio-femoral joint: (FLE-EXT and ADD-ABD);
▪ 86 muscles.

▪ Scaling the model
▪ Inverse Kinematics → Joint kinematics
▪ Inverse Dynamics → Joint moments
▪ Static Optimization → Muscles Forces
▪ Joint Reaction Analysis → Knee Contact Forces

SPSS

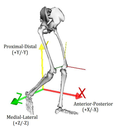
▪ ANCOVA with Sidak post hoc used to compare the peaks in KCF considering speed as covariate;
▪ Significance: $p \leq 0.05$

RESULTS

Using the gait speed as covariate, the 1st peak in total KCF was significantly higher (p = 0.047) in the OA2 comparing to the OA1. Similar changes were reflected in the PD component of the KCF, confirming a significantly increased 1st peak in OA2 compared to OA1(p = 0.043). The 2nd peak in the medial-lateral component was also significantly higher for OA2 than for C0 (p = 0.028), but not the first peak.

Table 1 - Peak total KCFs normalized to BW and gait speed with indication (*) of significant differences between groups.

GROUP	PEAK 1 (mean±SD)	PEAK 2 (mean±SD)	SPEED (m.s^{-2})
C0	3.990 ± 0.772	3.648 ± 0.674	1.232
OA1	4.070* ± 0.957	3.524 ± 0.601	1.294
OA2	4.458* ± 1.044	3.548 ± 0.590	1.209



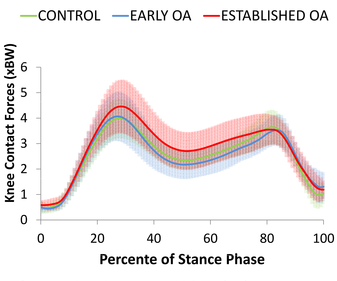


Figure 1 - Average KCF during stance phase across varying OA severities.

For OA2, total KCF remained elevated throughout mid stance compared to C0 and OA1 but lower values were observed in the 2nd peak reaching values similar to those reached by the OA1. Both were however lower than those found in the C0.

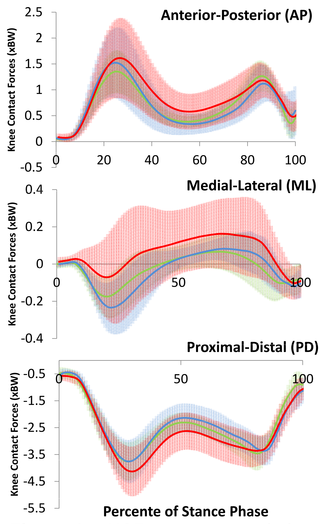


Figure 2 - KCF components during stance phase across varying OA severities.

CONCLUSION

▪ Despite a reduction in self-selected walking speed, total KCF were elevated in people with established OA except in the terminal stance. This finding suggests that excessive knee joint loading is a contributing factor to the progression of knee OA.

▪ Compressive but not medial shear forces were increased in the OA2 indicating that the overloading does mainly result from strategies that aim to stabilize the joint. Lateral forces were higher during the majority of stance in these patients, indicative of an unloading strategy of the medial compartment loading.

▪ KCF could not discriminate early OA patients and C0 subjects. This indicates that knee joint overloading may only become a contributing factor in the further progression of the disease. Therefore, an early diagnosis and subsequent therapeutic intervention should be directed to maintaining and stabilizing the loading conditions, therefore minimizing the role of increased mechanical loading as contributor to disease progression.

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